itic, and there was apparent paralysis of both legs. November 13 the nationt died. No autopsy was permitted.

Summary: Duration: Fourteen months previous to treatment.

Microscopie diagnosis: embryonal careinoma of the ehest of unknown origin. (A type of tumor which is unique in medical literature.) A large tumor mass almost filled the left chest. A hard movable tumor mass presented on elest wall. No metastases demonstrable. Marked dyspnea. Patient received 28 massive roentgen treatments (840X) between January 22, 1915, and August 1. 1915, during which time she became symptom-free and much improved in health The external tumor disappeared; the intrathoracie tumor diminished as shown in the illustrations. One month later the patient's symptoms suddenly returned and progressed despite all forms of treatment. Patient died of exhaustion.

BIHLIOGRAPHY.

- 1. Heineke: Mitteilung aus den Grenzgebieten der Medicin und Chirurgie xiv. 1905.

 - Menetrier and Touraine: Arch. d. mal. du coeur, 1910, iii, pp. 611-663.
 Heineke: München med. Wehnschr., 1903-1901.
 Rudhere: Arch. f. Auat. u. Physiol., 1907. Anat. Abiz. Supp., p. 123.
 Aubertin et Bordet: Centralld. f. micro. Medizin, 1909.
 - 6. Pigache et Beclere; Ref. Ztsehr, f. Röntgenk., 1913, xv, p. 2. (Bulletin de la
- Soc. nnatomique, 1911). 7. Regand et Cremien: C. R. Soc. de biol., 1911-1912, lxxii, p. 523. (Arch.
- d'Elect. Méd., 1912, xx, p. 481; 1912, xxi, pp. 144, 597). 8. Eggers: Ztschr. f. Röntgenk., 1913, xv, pp. 1, 44.
 - 9. Bergonie et Tribondeau: Arch. d'Elect. Méd., xiii, pp. 149, 474 et seq.; C. R.
- Soc. biol., 1904, v, p. 400 et seq.; ibid., December 10, 1906.
 10. Halberstadter: Berl. kliu. Wehnselr., No. 3, 1905.
- 11. Bergonie: Tribondeau and Recamier, Comp. rend. Soc. de biol., 1905, ibid., p. 284,
 - 12. Routier: Compt. rend. Soc. do biol., August, 1906.
 - 13. Specht: Arch. de Gynäkol., 1906, lxxviii, Heft 3.
 - 14. Okintschitz: Ref. Ztschr. f. Röutgenk., 1910, xii, p. 230.
 - 15. Saretzky: Ref. Ztschr. f. Röntgenk., 1910, xii, p. 235.
 - Langfelluer: München, med. Wehnschr., 1906. No. 44.
 Fraenkel: Centralbl. f. Gynäkol., 1907, No. 31.

 - 18. Krause and Zeigler: Fortschr. a. d. Geb. d. Röntgenstr., vol. x.

 - 19. Reifferschied: Ztschr. f. Röutgenk., 1910, Ie, p. 232.
 - 20. Simon: Zischr. f. Röntgenk., 1911, xiii, p. 371.
 - 21. Gassman: Fortschr. a. d. Geb. d. Röntgenstr., 1899, xi, p. 197.
 - 22. Leon: Cong. d. dent. dermat. Gesellsch., 1901.

SYPHILITIC AORTITIS.1

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The development of internal lesions in latent syphilis is attended by such vague indefinite clinical manifestations as to render diagnosis

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during their incipiency often a matter of extreme difficulty. This obscority of early signs and symptoms characterizes nortitis, and previous to the introduction of the Wassermann test and the employment of roentgen diagnostic technic, the lesion osnally came to light only when it had advanced to the production of gross and often obvious mechanical manifestations. On account of its usual prolonged latency it was described according to the elassification formerly in vogoe as a form of so-called parasyphilis, the occasional case that pursued an active rapid course hardly sufficing to distorb As a parasyphilitic manifestation, aortic syphilis this rating. remained more or less separated in interest from the parent disease, and the role assigned to it being that of a late mutritive secondary it was viewed with comparative indifference. The isolation of spirochetes in the tonics of diseased aortic and accumolated studies with the Wassermann test have in recent years combined to endow gortic syphilis with new significance, revealing the lesion in its proper light as troe syphilis sobject to all the activities and amenable to the same therapeotic attack as other forms of internal syphilis. Although pathologists describe fairly acote inflammatory changes at the root of the aorta in certain acote infectious, notably rhenmatism, these occur almost always in association with endocarditis, and, moreover, manifest a tendency to spontaneous subsidence very different from the progressive character of the nortitis of syphilis. These transitory non-specific types of nortitis are very little familiar to the clinician, so much so that it may be said, speaking elinically, there is but one form of aortitis, the syphilitic.

The Wassermann test has materially altered our views regarding the specific nature of aortic lesions. A positive Wassermann reaction has been obtained in over 80 per cent, of cases that showed characteristic aortic changes at aotonsy, Eich's statistics showing SLS per cent, and Pearce's 78.5 per cent. Citron used the Wassermann test in all chronic cases of aortic regorgitation and found it positive in 60 per cent. Collins and Sachs tested 13 cases of aortic valvolar disease finding the Wassermann positive in 11. By way of contrast they record an equal number of eases of porely mitral disease similarly tested, with Wassermann positive in but 2 instances. Still more direct evidence of the syphilitic nature of aortic disease has been furnished by the isolation of the spirochete of syphilis in the tonics of diseased norte, such a finding being now of common occorrence. Moreover, the fact that lesions practically the same as syphilitic nortitis in the acquired disease have been found to exist in a high percentage of infants with congenital syphilis affords strong sopport to the claim that the lesion in adolts is doe to syphilis.

Syphilitie aortitis before it involves the valves is often latent. After it has produced aortic valve incompetency its progress is rapid, often bringing life to an end within two or three years.

Aortic regurgitations of syphilitic origin do very badly probably because of the association of deformed valves and constricted coronary orifices. The prognosis is far more serious than in cases of rhemnatic origin even when of equal degree, compensation being less well established and the norta more diluted.

Early in the course of aortic syphilis the involvement of a limited portion of the artery may appear of slight immediate significance. Its progression involves the risk of serious damage with, it may be, dilatation or ancurysm angina pectoris or most commonly aortic regurgitation. To detect this insidious lesion before it has advanced to the production of mechanical secondaries is consequently of great importance. Clinical recognition of the disease in its earliest stages gains greatly in importance from the fact that it may be treated by intensive antisyphilitic methods with some prospect of control. The diagnosis is often extremely difficult, since limited and circumscribed changes in the ascending portion of the aorta without involvement of the valve ring are seldom accompanied by clearly defined symptoms or signs. The greatest difficulty is encountered in individuals not recognized as being syphilitie. It is often the case for aortitis to be the only manifestation of the disease in otherwise latent syphilis, and one may expect in a considerable percentage of cases to encounter a completely negative history of syphilis. In one-half of Benry's cases and in 28.9 per cent, of Symmer's natopsy material, aortitis was the only manifestation of the disease detected at autopsy. The majority of patients coming under clinical recognition for aortic syphilis are in middle life, and experienced clinicians understand the significance of angina pectoris and aortic regargitation originating in the adult.

Our most valuable aids in the diagnosis of obscure aortic disease are the Wassermann test and rocutgen-ray examination.

It is now generally conceded that a positive Wassermann reaction means active synhilis somewhere in the body. From the frequency with which syphilitie arotitis is found aecidentally at autopsy (according to Eich 42.8 per cent. of cases) we may assume that the infection may be harbored in that part of the vascular system for years without detection; indeed, it might even appear that the aorta is one of the main situations in which the infection may lurk in latent syphilis. A persistent Wassermann reaction in an otherwise latent ease may consequently direct suspicion toward the norta and a roentgen-ray study of this area should be made. The absence of a definite or confessed history of syphilitie infection should not be allowed to divert attention from such a possibility. This is especially important in women in whom historical data on this point arc, for obvious reasons, obscure and unreliable. negative blood Wassermann in a known syphilitic should not be accepted as inconsistent with a possible aortic lesion. Progressive aortitis may exist, although the blood has been Wassermann negative for years. The luctin test of Noguchi may occasionally reveal the disease when the Wassermann reaction is negative, as in certain cases presently to be related. Bearing in mind this frequency of aortitis in latent lucs it is well that we should consider the possibility of a syphilitic etiology in any case of cardiovascular degeneration of obscure origin. If serological studies confirm this suspicion the aorta should be carefully examined by means of modern roentgenray technic.

The symptoms of syphilis of the aorta are pain in and about the precordium, dyspnea, hoarseness, paroxysmal cough, tachycardia, arrhythmia, general weakness and lassitude, and, under certain circumstances, clevated blood-pressure. These symptoms are seldom clearly defined during the period of early development of the disease. Various clinical criteria, such as the shape of the heart to percussion, peculiarities of rhythm, the paroxysmal nature of cardiac symptoms have been proposed to identify syphilitic aortitis. One need not emphasize the unreliability of such data.

There are certain physical signs which may be elicited by examination in most well-developed cases, but they are not of such a character as to especially indicate aortic syphilis. Among the most significant of these signs are a roughening or impurity of the first aortic sound and u sharp ringing aortic second tone, which if the aorta is much dilated may have a bell-like quality. Extension upward of percussion dulness over the sternum may exist, and there may be abnormal pulsation in the carotids and subclavian with an impulse in the episternal notch. None of these signs are, however, sufficiently characteristic to furnish grounds for diagnosis of syphilitic aorticis, since they are frequently noted in aortic atheroma and high blood-pressure states not connected with syphilis.

The primary aortic involvement is a small cell or gramulomatous infiltration of the tunica media (mesaortitis) along the course of the vasovasorum. Eventually all three of the main coats of the aorta become affected the intima by secondary proliferative thickening. Congulation necrosis, and subsequent connective-tissue changes lead to slow but extensive damage to the vessel wall, so that fairly characteristic gross appearances become established. Although maximum involvement appears to fall upon the first part of the arch and the ascending aorta probably because of the greater functional strain to which this portion of the vessel is subjected, no section throughout the vessel is exempt from attack. The involved areas appear as more or less well-defined elevated patches, the surface of the larger areas being often extremely irregular, with pittings, sears, and puckerings, making what Allbutt describes as "knotty masses" in the vessel wall (Fig. 1).

When secondary changes are extensive they are characterized not by calcification, as in arteriosclerosis, but by a gradual corrosion or thinning by sear formation, so that the vessel wall may become translucent at points and there may be tiny aneurysmal bulgings or possibly a true saccular aneurysm. Frequently cellular infiltration about the months of the branches of the arch may cause distinct narrowing of innominate and left carotid or more or less occlusion of the coronaries.

In the earlier stages the vessel is perhaps but little dilated or deformed. If viewed from without little change may be noted (Fig. 2). It is on the inner surface of the artery that the changes appear (Fig. 3). The tendency of the disease in development is to spread to the aortie valve and by a general periarteritis in the eusps and between them the semilunar valve flaps become thickened and deformed, giving rise to valve incompetency. It is a common



Fig. 1.—Syphilitic norlitis with diffusely dilated vessel. (From Pathological Museum, Rush Medical College.)

and important clinical observation that the aorta may be gravely affected, although the peripheral arteries apparently remain normal, and Brooks has emphasized the fact that the coronaries are apt to show a degree of involvement out of all proportion to general arterial change. It is the seat of the disease in the suprasigmoid portion of the aortic arch that threatens the integrity of the coronaries, and the steady progression of the disease accounts for the nortic valve defects aneurysm and anginas which constitute the morbid end-products of syphilitic nortitis.

Of all the methods of diagnosis to determine the presence of aortic disease, the roentgen ray is the most valuable. By its aid we may detect slight changes in the caliber and shape of the vessel which entirely clude the methods of physical examination. Both fluoroscopic inspection and plate studies should be employed, the



Fig. 2.-Syphilitic nortitis; outer surface of vessel.



Fig. 3.—Syphilitic aartitis; inner surface of vessel. (From Pathological Museum of Rush Medical College.)

former for preliminary observation and the detection of abnormal pulsation and the latter to secure definite outlines and measurements. The various roentgen-ray plates berewith reproduced were taken in the standing position, with a few seconds' exposure, and focal distance of seven feet, which provides for practically parallel rays and obviates distortion of the shadow.

The shadow of the normal aorta in the roentgenogram (Fig. 4) lies in its first portion behind the stermm and does not project beyond the right aurienlar curve. At the arch where it curves backward and to the left there appears a more or less well-rounded knob seen to the left of the stermm well above and distinct from the cardiac shadow. This aortic knob is not equally well defined in all normal nortic. In the asthenic or dropped heart type it may hardly show at all, whereas in occasional cardiac roentgenograms it may be so prominent a feature as to be mistaken for anencysmal bulging.



Fig. 4.-Normal aortic shadow.

It varies in prominence according to age, being less defined in early life than in adults, and in the aged and arterioselerotic individuals the shadow may be sharply outlined and intensilied from selerous thickening and lime deposits (Fig. 5). It is less apt to be prominent in adult females than in males of corresponding age.

In a ortitis the roentgen ray reveals two kinds of changes, $i.\ c.$, alterations in density of shadow and changes in contour. In early a ortitis the pathological process is almost invariably conlined to the very origin of the norta. As the vessel is not at this stage thickened and no lime deposits exist there may be merely a slight dilatation of the lirst part of the arch with exaggerated pulsation at that point. Wessler points out that some dilatation of the aorta is a very common finding in routine cardiac roentgenography, so that little special significance attaches to it. It is practically con-

stant in ordinary sclerosis of the aorta and in high blood-pressure states. In non-specific aortic sclerosis, however, it is nearly always accompanied by a prominence of the whole arch, and if bloodpressure be elevated by hypertrophy of the left ventricle.

In early cases of aortitis the heart is rarely enlarged unless there is coexisting valve defect or hypertension. Of course, aortitis and atherona may be combined in the same case, when computation will be rendered difficult. With the progress of aortitis the artery becomes more and more dilated, and, as a result, the aortie shadow as it ascends from the heart is widened so that it projects beyond the shadow of the curved right anricle. In most instances the shadow is regular, but it may show slight bulgings or protrusion.



Fig. 5.-Non-specific acrtic selerosis. Note lime deposits at acrtic knob.

Should the nortic dilatation extend to the arch the knob as it curves to the left may lose its well-defined curve, becoming flattened and broadened. Longcope has described a type of case in which the nortic shadow appears as a more or less truncated cone, with rounded apex and broad base resting on and fusing with the cardiac shadow. In any well-developed instance there may appear above the nortic shadow a pyramid-shaped shadow with base upward and beneath the clavicles. This seems to represent the dilated great arteries as they arise from the arch (Fig. 6).

While in the earlier stages of nortitis roentgen evidence may consist merely of a slightly increased curvature and pulsation at the nortic ampulla, a gradual dilatation and lengthening of the vessel supervene with further progress of the disease. These changes combine to broaden and elevate the aortic shadow, and there occurs in most cases some culargement in the size of the heart whether



Fig. 6.—Advanced syphilitic aortitis with nortic valve insufficiency. Marked dilatation of aortic arch and great vessels as they arise from arch.



Fig. 7.—Non-syphilitic nortic insufficiency illustrating "nortic configuration" of heart shadow

aortie insufficiency be present or not. The increase in cardiac diameters affects to a much greater degree the long diameter than

the broad diameter of the heart, the organ assuming a position more nearly horizontal than normal, producing a heart shadow somewhat boot-shaped, the heart lying low on the diaphragm. This type of heart shadow is very similar to that seen in high blood-pressure states (nephritis), although the marked pulsation of the left ventricle seen in hypertension is less manifest. This type of heart might be termed the aortic heart (aortic configuration) (Fig. 7) in contradistinction to the broad, rather round, or bag-shaped heart of mitral disease ("mitral configuration") (Fig. 8). With the spread of the disease to the aortic valve and the production of aortic insufficiency we find the heart shadow much augmented and its horizontal configuration correspondingly magnified. In advanced cases with superadded relative mitral insufficiency both



Fig. S.-Mitral disease illustrating "mitral configuration" of heart shadow.

broad and long diameters are increased, combining to produce a very bulky heart shadow, the true cor boyis (Fig. 9).

It is obvious that until there is some dilatation, thickening, alteration of curve, or abnormal pulsation in the vessel, roentgen diagnosis of aortic disease is impossible. In consequence of this fact the roentgen ray cannot be relied upon for its detection in the early stage before mechanical defects begin to make their appearance. Clinical considerations and the Wassermann test must establish the diagnosis at this stage. An illustrative case may be eited.

Case I.—A colored waiter, aged twenty-one years, complained of persistent aching pain in the left shoulder of two months' duration. This was accompanied by a recurrent substernal soreness, pulpi-

tation, and a dry paroxysmal cough. He gave a history of chance two years previous to consultation. On examination an appreciable enlargement of the heart could be made out, and along the left border of the stermm a faint diastolic whiff was heard. Firm pressure over the manubrium sterni caused a deep-scated aching soreness. Blood Wassermann positive. Blood-pressure systolic, 155; diastolic, 85; pulse-pressure, 70. The roentgen ray beyond some enlargement of the heart failed to reveal anything of significance.

The clinical diagnosis was manifestly aortitis of rather active type, yet at a stage too early for stretching of the aorta or other mechanical defect to appear upon the roentgen-ray plate.



Fig. 9.—Extreme cardiac effects of aortic syphils, i. c., aortic regargitation, relative mitral insufficiency, pulmonary hypo-tasis. This patient is alive, two years after this plate was taken.

In contrast to this type of case may be cited the following instance in which the roentgen ray confirmed and established beyond doubt the clinical diagnosis.

Case II.—A white man, aged forty years; widower; one child; complaint: hoarseness, cough on exertion, dyspuca. Acknowledges heetic infection ten years previous to consultation, for which he received almost continuous treatment for four years. Blood Wassermann, typically positive. Physical examination showed a somewhat enlarged heart, with a ringing second aortic tone. The pulse was regular. Blood-pressure: systolic, 140; diastolic, 100; pulse-pressure. 40.

The roentgenogram (Fig. 10) showed typical aortic configuration of the heart, with bulging of the first part of the ascending aorta,

The following example of well-developed syphilitie acritis possesses points of musual interest:

Case III.—Single white man, aged forty years; complaint, palpitation and irregular heart action, lack of endurance, lassitude, and depression. Gave a history of lines at age of twenty-nine, with continuous treatment for two years. For three years previous to consultation blood Wassermann tests have been made at regular six-month intervals, with invariable negative reports. The last Wassermann test was taken six months before roentgenogram was made and proved negative.



Fig. 10.—Case II. Heart shadow displays agrife configuration with bulging of first portion of ascending agria.

Physical examination revealed an enlarged heart, aortic systolic murnur, accented second aortic tone, premature contraction arrhythmia. Blood-pressure: systolic, 210; diastolic, 150. Early tabetic signs. The nrine contained albumin and casts, Renal function index with the 'phthalein test 60 per cent. Clinical diagnosis: syphilitic inyocarditis, nephritis. The roentgenogram showed a typical nortic type of heart and stretched aorta.

The points of particular clinical interest in this case are the existence of extensive and probably progressive cardiovascular lesions of syphilitic type in an individual who for several years had been Wassermann negative, and was led to consider himself cured of his syphilis. The extent of damage already wrought by the cardiovascular disease would appear to indicate that it was of some years' standing.

The peculiar lassitude and depression which was the chief complaint of this patient is a symptom interpreted by Allbutt as significant of coronary involvement. A point of interest to be noted in this case is the association of aortic disease with signs of early tabes. This is an association frequently noted in the literature. In manifest tabes, Stadler found aortic disease in almost all cases. He regarded so-called cardiae crises as being in reality angian pectoris due to coronary stenosis. Strümpell emphasized the frequent association of aortic syphilis and tabes and statistics from various sources attest its frequence. In general, symptoms of aortic disease appear later than the earliest tabetic manifestations. This is perhaps not because aortitis is a later development but rather that it has a longer latency than tabes.

The cases thus far briefly described represent aortic disease in a relatively early stage of its development. Progress of the lesion inevitably tends to impair the integrity of the aortic valve producing as a result insufficiency of the valve apparatus. How soon this mechanical secondary comes to pass depends more upon the activity of the process than upon its duration. No estimate can be given of the average period required for the production of valvular defects, although, generally speaking, it is certainly not an early development. Occasionally valvular disease is seen to develop early and run a rapid fatal caurse, as in the following instance:



Fig. 11.—Case IV. Aortic and cardiac didatation.

Case IV.—Colored porter, aged twenty-five years; complaint, severe dyspnea, precordial pains. Duration of symptoms, six weeks; advent sudden; previous health considered good until development of foregoing symptoms. Luctic infection at age of

twenty; no active antisyphilitic treatment. Blood Wassermann strongly positive. Clinical diagnosis: aortic insufficiency, relative mitral insufficiency, pulmonary hypostasis. Termination, death after one week in hospital seven weeks after development of cardiac symptoms. Roentgenogram (Fig. 11) was taken eighteen hours before death and shows a greatly enlarged heart increased in hoth long and broad diameters. The aortic shadow is increased in hreadth and elevated reaching as high as the sternoclavicular angle. This case illustrates how rapid may be the cardiac disorganization produced by aortitis, death resulting in this case five years after the initial syphilitie sore.

Aortic disease was formerly looked upon as a late development in syphilis. While, generally speaking, and especially in a clinical sense, this may perhaps he true, it is by no means always so. Along with other cases reported in recent contributions (Brooks, Longcope) the foregoing clinical history serves to discount the claim that aortic disease has a long latency and slow progression.

The following case represents an interesting variant of the usual elinical course of aortic disease:



Fig. 12.—Case V. Anterior front view. Increase in both long and broad diameters; dilated north.

Case V.—A white engineer, aged thirty-two years; married; two children, one healthy, one, dead at three weeks, "had a patent foramen ovale." Syphilis denied. Complaint: dyspuea, cough, tachycardia. Duration of symptoms three months; advent sudden, following exertion at high altitude. Urine contains albumin and easts. Renal function index, 50 per cent. Blood-pressure: systolic,

200; diastolie, 80; pulse-pressure, 120; blood Wassermann, strongly positive. Clinical diagnosis: aortic insufficiency, mitral insufficiency, nephritis. Roentgenogram (Fig. 12) shows the heart shadow increased in both length and breadth from the combined aortic and mitral incompetency. The shadow of the aorta is widened and rises high in the chest. Another semilateral roentgen exposure (Fig. 13) shows this well and indicates beautifully the enormous thickness of the heart shadow.

The duration of this patient's syphilis must rest on assumption and may perhaps be estimated as less than seven years (the age of his healthy child) and more than four (the date of birth of the congenitally defective child). The development of nephritis may by increasing nortic and cardiac strain have served to hasten the progress of the nortic disease.



Fig. 13.-Case V. Semilateral view.

Case VI.—Colored cook, aged thirty-six years; complaint: dyspanea, chronic congh, orthopnea, slight edema. Duration of symptoms, six months. Previous health good. Syphilis denied. Blood Wassermann, strongly positive. Clinical diagnosis, nortic regargitation. Roentgenogram showed nortic conliguration increase in cardiac diameter 32 per cent. This case is a typical one of progressive syphilitic nortitis in latent syphilis and demonstrates how silent may be the progress of the vascular lesion until grave valvular secondaries arise. The liability of the colored race to cardiovasenlar syphilis is notorious and need not be emphasized here.

Syphilitic aortitis may exist in individuals who fail to react to the Wassermann test, as is shown in the following case: Case VII.—Hebrew peddler, aged forty-nine years; married; three children; complaint: pains in body, dyspnea, cough, general weakness. Duration of symptoms, one year. Syphilis denied. Blood and spinal fluid Wassermann negative. Luctin test typically positive. Roentgenogram (Fig. 14) shows heart to be of aortic configuration; type asthenique; dilated and lengthened aorta. Increase in cardiae diameter 10 per cent.



Fig. 14.—Case VII. Increase in cardiac diameter 10 per cent. Heart is of acrtic configuration; asthenic or dropped heart type.

This with the following case illustrate that a negative Wassermann both blood and spinal fluid tests should not be taken as indubitably disproving a syphilitie etiology for aortic disease. The luctin test of Noguchi may reveal the disease and supply the necessary diagnostic link.

Case VIII.—Colored honsemaid, aged thirty-eight years; widow; complaint: orthopnea, chronic productive cough, no edema. Duration of dyspnea one week, cough chronic for three years. Syphilis denied. Blood and spinal fluid Wassermann both negative. Luctin test typical pustular reaction. Roentgenogram shows heart to be of aortic shape; costal-phrenic angle not clear on plate; dilated aorta. Increase of cardiac dianyeter 20 per cent.

During residence in hospital patient had six typical attacks of angina pectoris, the paroxysms being relieved by nitrites. Intensive treatment with neosalvarsan and mercury brought about great subjective improvement. Three months after leaving hospital there had been no recurrence of angina paroxysms.

The following 2 cases represent slightly different aspects of aortic syphilis. In both instances the activity of the disease appears to fall on the further distribution of the vessel. In the first of the vol. 154, No. 1.—JULY, 1917.

2 cases the transverse portion of the arch and in the second the abdominal aorta had borne the brunt of the disease with the formation of aneurysm in both instances.



Fig. 15.-Case IX. Front view, showing prominent nortic knob only.



Fig. 16.—Case IX. Semilateral view, showing aneurysm of posterior aspect of aortic arch.

Case IX.—Colored porter, aged thirty-four years; single; complaint: severe brassey unproductive cough, paroxysmal dyspnea, voice husky. Duration six months. Physical examination: slight tracheal tug, obscure systolic murmur at inner edge of left scapula just above its angle. Heart and chest otherwise negative. Left vocal cord paresis. Leutic infection at twenty-four. Blood Wasser-

mann strongly positive. Clinical diagnosis: aneurysm of thoracic aorta. Roentgenogram: the customary anterior-front view of the heart (Fig. 15) revealed a decided increase in the prominence of the aortic knob but the aortic and heart shadows otherwise were normal. As this somewhat obtrusive prominence of the knob is not rare in the normal aorta, and less rare in aortic atheroma, it could not be interpreted as of diagnostic value. An anterior semi-lateral view of the aortic shadow (Fig. 16) solved the problem, revealing a well-marked aneurysmal dilatation of posterior aspect of the aorta as it curves to the left and downward. This bulging could be seen rather indistinctly with the sercen, but appeared much more clear-cut on the plate. The case furnishes a good example of the importance of screening all cases and of taking plates of more than one aspect of the aorta when doubt exists.



Fig. 17.—Case X. Note subcardiac shadow corresponding to alxiominal ancurysm.

Case X.—Colored porter, aged twenty-five years; complaint: pain in right hip; duration two weeks; previous health satisfactory. Physical examination revealed a plainly visible mass in epigastrium pulsating with expansile thrust synchronously with the heart's action. Luctic infection at sixteen. Blood Wassermann strongly positive.

Fluoroscopic examination, the stomach being distended with barium sulphate suspension, showed the stomach displaced to the left and pulsation of lesser curvature in contact with ancurysm.

Roentgenogram (Fig. 17) shows a practically normal heart and aortic shadow; below the heart is seen a dark shadow corresponding to the ancurvsm.

The progress of the case was marked by gradual enlargement of the epigastric pulsating tumor and the formation of a large mass in the upper right abdomen, displacing the liver and right kidney downward and outward into the flank with the erosion of lower ribs posteriorly and the formation of a soft pulsating tumor in that region. Death occurred suddenly three months after admission.



Fig. 18.—Case X. Roentgenogram taken just before death. Note displacement of right displacage with compression of right lung and displacement of heart upward. Aorta dilated.

Antopsy: Ventral sacenlar anenrysm of the abdominal aorta with the formation of a large false ancurvsm, which contains organized blood clot weighing 990 grams. This false aneurysm is adherent to the diaphragm and right lower lobe of hing and to the eroded ribs and vertebrae. It extends down as far as the right psoas muscle. This mass is ruptured through the diaphragm near the aortic opening into the right pleural sac. A second roentgenogram (Fig. 18) was taken shortly before the patient's death. A comparison of the two roentgenograms will reveal a considerable contrast in locart contour. Upon admission the cardiac sounds were clear and heart outlines little different from normal. Toward the termination of the case the diastolic murmur of aortic insulliciency became plainly audible and the cardiac diameters increased. It is interesting to record that at autopsy no defect of the nortic valve cusps was apparent, but that the aortic ring was obviously dilated. Reference may be found in the literature to the fact that although the physical signs of aortic regnigitation exists during life, at necropsy the valves may appear surprisingly normal, regurgitation apparently depending upon dilatation of the cortic ring.